2.
From epidemiological studies a very consistent picture of an elevation of about 30 % in risk (for both death and non-fatal coronary events) has emerged, even after controlling for other known cardiovascular disease risk factors (7). These population studies are, in fact, more consistent in their findings than even the studies demonstrating that passive smoking causes lung cancer.

page 3

7. Wells, A.J., Passive smoking as a cause of heart disease, J. Am. Coll. Cardi 1994; 24:546-554

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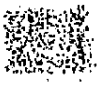

3.
....., the presence of the dose-response relationship in many studies (10), and the fact that active smoking causes heart disease in smokers, would be enough to conclude that passive smoking causes heart disease in nonsmokers and to proceed with a quantitative risk assessment.

page 3

10. Wu-Williams, A. Samet, J., Environmental tobacco smoke: Exposure-response relationships in epidemiologic studies. Risk Analysis 1990; 10:39-48

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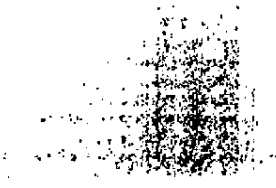

4.
If we moved to Denver - a mile high - where there is less oxygen, our bodies would adapt by adjusting hematocrit levels, the fraction of blood occupied by oxygen-carrying red blood cells. The body would increase the hematocrit in order to expand the oxygen carrying capacity of the blood as a compensation for the lower oxygen in the air.

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5.

People who smoke cigarettes are chronically and continually attacking their cardiovascular systems by reducing the oxygen-carrying capacity of blood (because of the carbon monoxide in the smoke) and by increasing the demands on the heart muscle itself because nicotine increases heart rate .....

page 5

12. Benowitz, N.L., Nicotine and coronary heart disease. Trends Cardiovasc Med 1991;1:315-321

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6.
..... first, nonsmokers' hearts and vascular systems have not attempted to adapt to the acute poisoning from the chemicals in the secondhand smoke.

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7.
Second, it appears that the cardiovascular system is extremely sensitive to many of the chemicals in secondhand smoke and that smokers may have achieved the amximum response possible to at least some of the toxins in the smoke,.....

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8.
The qualitative differences between the effects of ETS on smokers and nonsmokers explains the relatively high relative risks associated with passive smoking compared to active smoking, even though passive smokers absorb much smaller doses of the toxins in cigarette smoke than the smokers do (10).

page 5.

10. Wu-Williams, A. Samet, J., Environmental tobacco smoke: Exposure-response relationships in epidemiologic studies. Risk Analysis 1990; 10:39-48

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9:
....., the smoke from one cigarette is enough to produce substantial effects on the cardiovascular system.

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10.
The availibility of thes animal models has contributed significantly to our understanding of the precise mechanims by which passive smoking damages the heart.

page 6

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11.
Passive smoking reduces the ability of the blood to deliver oxygen to the heart muscle.

page 6

14. US Department of Health and Human Services. The Health Consequences of Smoking: Cardiovascular Disease. A Report of the Surgeon General. Public Health Service, Office on Smoking and Health, 1983. DHHS(PHS) 84-50204

15. US Department of Health and Human Services. The Health Consequences of Involuntary Smoking: Cardiovascular Disease. A Report of the Surgeon General. Office on Smoking and Health, Centers for Disease Control, Public Health Service, 1986. DHHS(CDC) 87-8398

16. Moskowitz, W., Mosteller, M., Schieken, R. et al., Lipoprotein and oxygen transport alterations in passive smoking preadolescent children: The MCV Irwin study. Circulation 1990; 81:586-592

17. US Environmental Protection Agency: Air quality criteria for carbon moxoide. US Environmental Protection Agency, 1990. USEPA Document No. EPA/600/8-90/043A

18. Leone, A., Mori, L., Bertanelli, F., Fabiano, P., Filippelli, M., Indoor passive smoking: Its effect on cardiac performance. Int. J. Cardiol. 1991; 33:247-252

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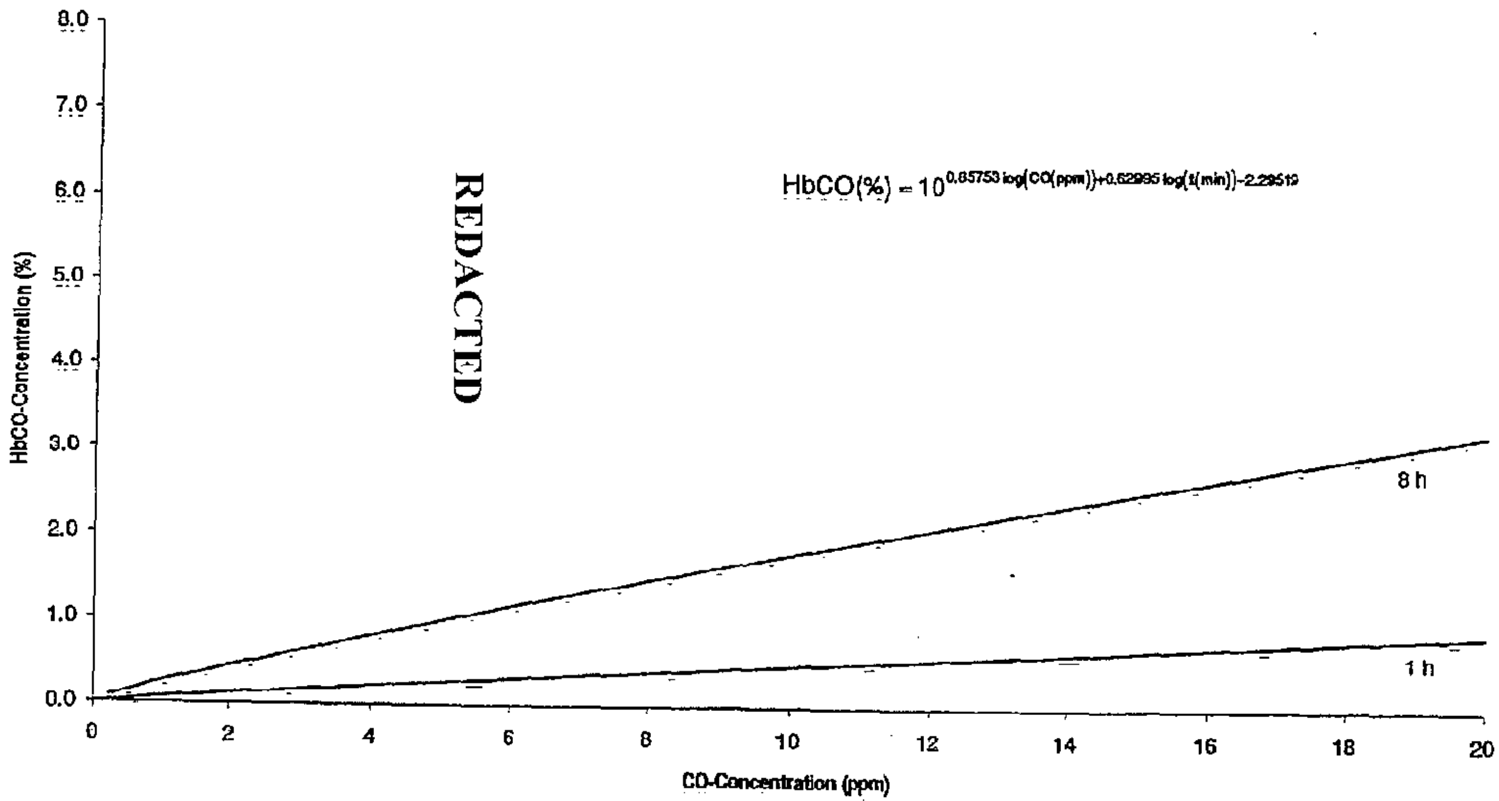

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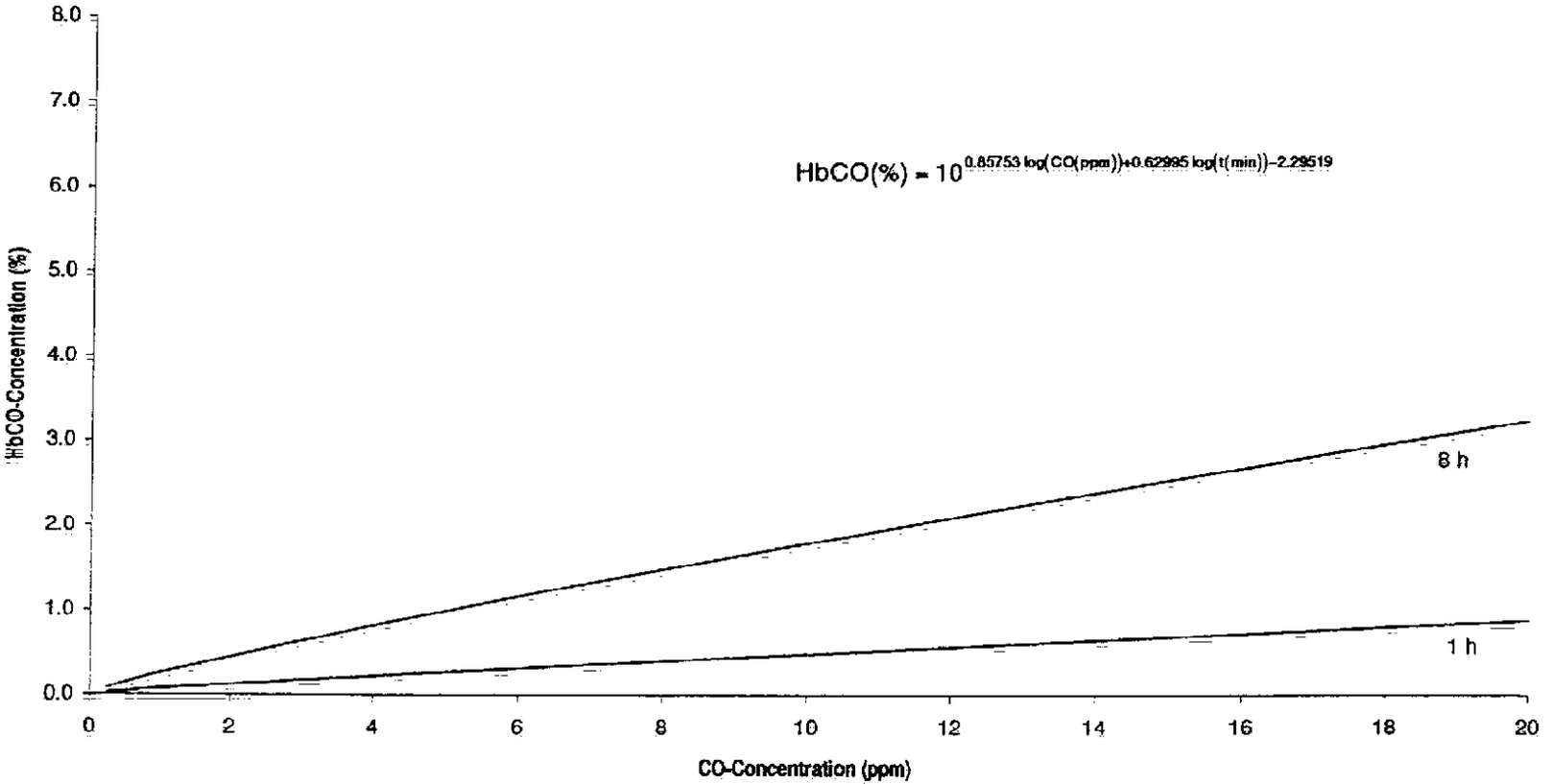

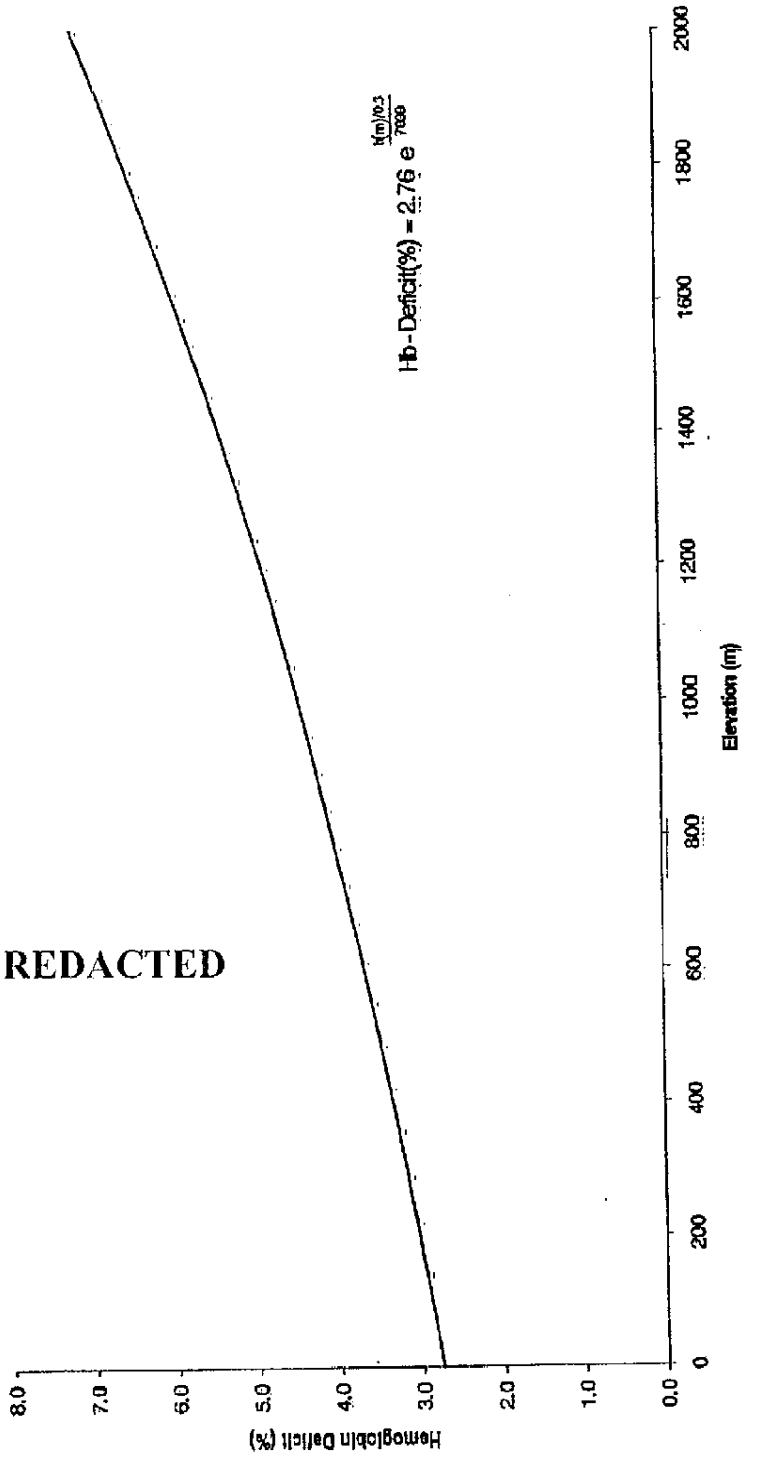

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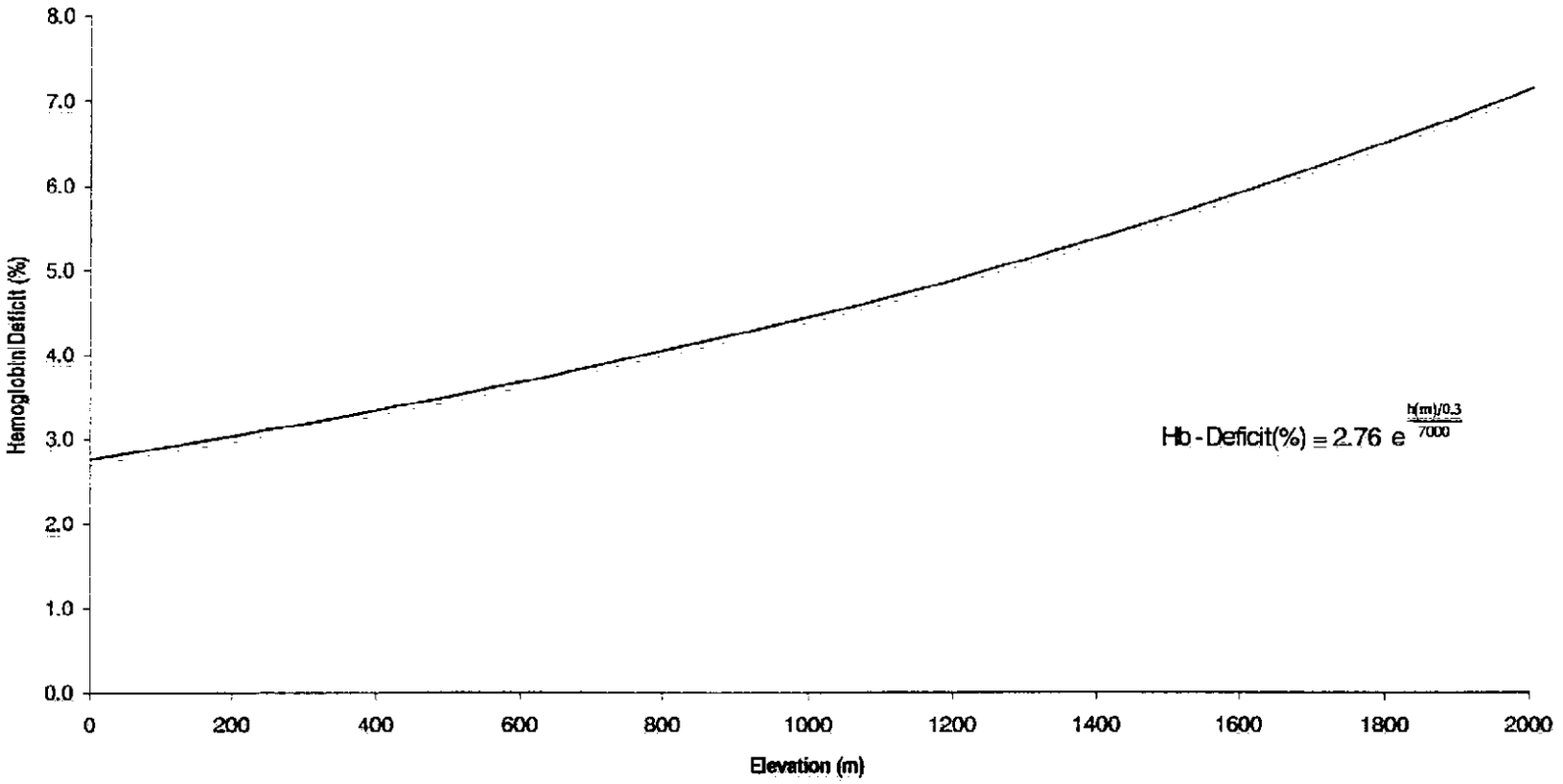

Height (m)	Hb (%)
0	2.76
100	2.89
300	3.18
600	3.67
1000	4.44
1300	5.13
1600	5.91
1900	6.82
2000	7.15

CO (ppm)	HbCO (%) Zeit (min)	
	60	480
0.25	0.02	0.08
1	0.07	0.25
5	0.27	0.98
10	0.48	1.78
15	0.68	2.53
20	0.87	3.23

12.
Even at the lower levels of carboxyhemoglobin observed in passive smokers, it can have an effect on exercise performance and how the heart beats, in people who already have heart disease. Children of smoking parents have elevated levels of a chemical called 2,3-diphosphoglycerate (DPG).

page 7

9. Gidding, S., Morgan, W., Perry, C., Isabel-Jones, J., Bricker, J.T., Active and passive tobacco exposure: A serious pediatric health problem. A statement from the Committee on Atherosclerosis and Hypertension in Children, Council on Cardiovascular Disease in the Young, American Heart Association. Circulation 1994; in press

16. Moskowitz, W., Mosteller, M., Schieken, R. et al., Lipoprotein and oxygen transport alterations in passive smoking preadolescent children: The MCV Irwin study. Circulation 1990; 81:586-592

19. Pomrehn, P., Hollanbush, J., Clarke, W., Lauer, R., Children's HDL-chol: The effects of tobacco, smoking, smokeless, and parental smoking. Circulation 1990; NEED CITATION

20. Moskowitz, W.B., Mosteller, M., Hewitt, J.K., Eaves, L.J., Nance, W.E., Schieken, R.M. Univariate genetic analysis of oxygen transport regulation in children: The Medical College of Virginia twin study. Pediatr. Res. 1993;33:645-648

21. Feldman, J., Shanker, I.R., Etzel, R.A. et al. Passive smoking alters lipid profiles in adolescents. Pediatrics 1991;88:259-264

13:
Studies have shown that these enzymes do not work as efficiently in rabbits exposed to the smoke from three cigarettes smoked over thirty minute period a single time, twice daily for two weeks, and twice daily for eight weeks. These are extremely brief exposures, even compared to the lifetime of a rabbit. Even a single exposure to environmental tobacco smoke reduced the rate at which oxygen was converted into ATP.

page 8

22. Gvozdjak, J., Gvozdjakova, A., Kucharska, J., Bada, V. The effect of smoking on myocardial metabolism. Czech Med 1987;10:47-53

14.
Thus, not only does breathing secondhand smoke reduce the ability of the blood to get oxygen to the heart muscle, but it also reduces the ability of the heart muscle to make effective use of the oxygen it does get (23).

page 8

23. Gvozdjakova, A., Kucharska, J., Gvozdjak, J. Effect of smoking on the oxidative processes of cardiomyocytes. cardiology 1992;1992:81-84

15.
Children of smoking parents have elevated levels of a chemical called 2,3-diphosphoglycerate (DPG). This chemical appears in red blood cells in an effort to modify how they handle oxygen to compensate for chronic oxygen starvation (9, 16, 19-21).

page 7

9. Gidding, S., Morgan, W., Perry, C., Isabel-Jones, J., Bricker, J.T., Active and passive tobacco exposure: A serious pediatric health problem. A statement from the Committee on Atherosclerosis and Hypertension in Children, Council on Cardiovascular Disease in the Young, American Heart Association. Circulation 1994; in press.

16. Moskowitz, W., Mosteller, M., Schieken, R. et al., Lipoprotein and oxygen transport alterations in passive smoking preadolescent children: The MCV Irwin study. Circulation 1990; 81:586-592

19. Pomrehn, P., Hollanbush, J., Clarke, W., Lauer, R., Children's HDL-chol: The effects of tobacco, smoking, smokeless, and parental smoking. Circulation 1990; NEED CITATION

20. Moskowitz, W.B., Mosteller, M., Hewitt, J.K., Eaves, L.J., Nance, W.E., Schieken, R.M. Univariate genetic analysis of oxygen transport regulation in children: The Medical College of Virginia twin study. Pediatr. Res. 1993;33:645-648

21. Feldman, J., Shanker, I.R., Etzell, R.A. et al. Passive smoking alters lipid profiles in adolescents. Pediatrics 1991;88:259-264

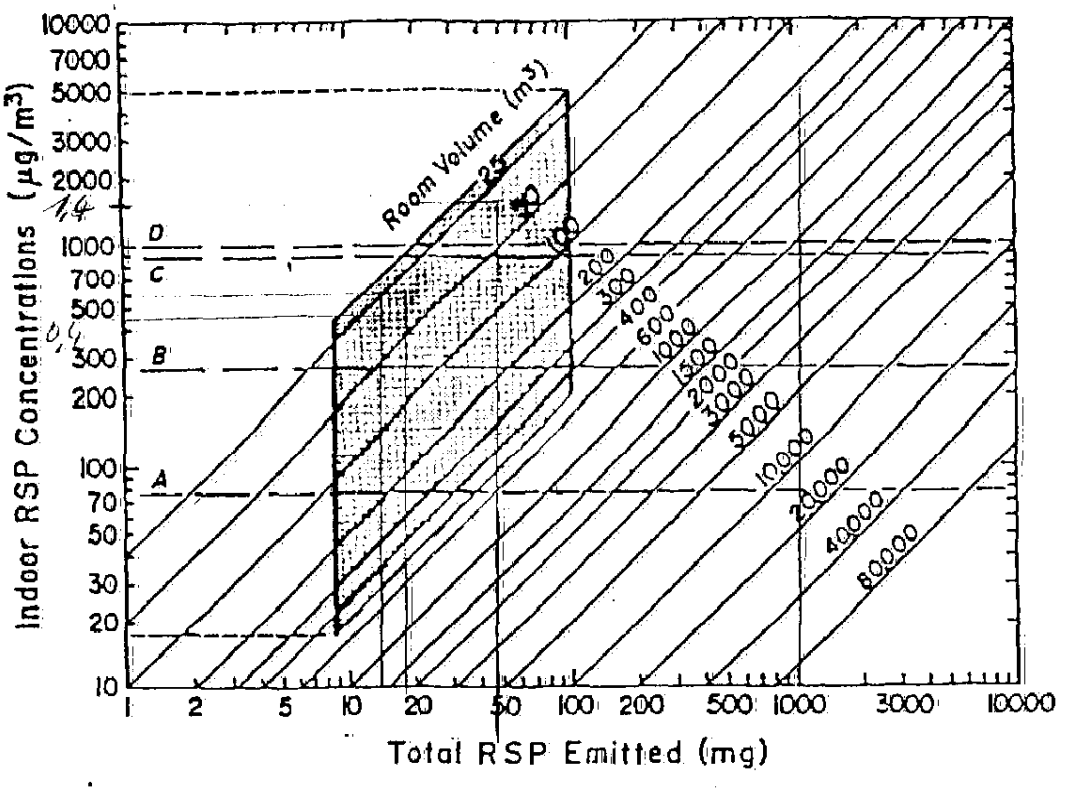

Figure 3-2. Diagram to calculate the ETS-associated respirable suspended particle mass (RSP) concentration in $\mu g/m^3$ in a space as a function of total mass of ETS-generated RSP emitted in mg (determined from Figure 3-1) and the volume of a space (diagonal lines). The concentrations shown assume a background level of zero in the space. The particle concentrations shown are estimates during smoking occupancy. The dashed horizontal lines (A, B, C, and D) refer to National Ambient Air Quality Standards (health-related) for total suspended particulates established by the U.S. Environmental Protection Agency. A is the annual geometric mean. B is the 24-hour value not to be exceeded more than once a year. C is the 24-hour air pollution emergency level. (D is the 24-hour significant harm level.) Shaded area shows the range of concentrations expected (from Figure 3-1) for a range of typical volumes of U.S. residences and rooms in these residences.

Source: NRC, 1986.

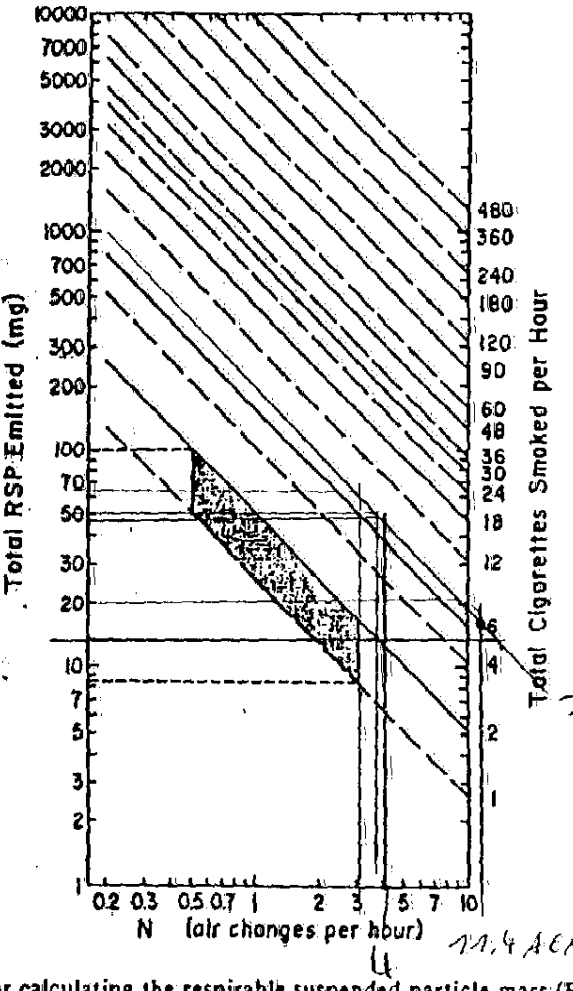

Figure 3-1. Diagram for calculating the respirable suspended particle mass (RSP) from ETS emitted into any occupied space as a function of the smoking rate and removal rate (N). The removal rate is equal to the sum of the ventilation or infiltration rate (n_v) and the removal rate by surfaces (n_s) times the mixing factor. The calculated ETS-related RSP mass determined from this figure serves as an input to Figure 3-2 to determine the ETS-related RSP mass concentration in any space in $\mu g/m^3$. Smoking rates (diagonal lines) are given as cigarettes smoked per hour. Mixing is determined as a fraction, and n_v and n_s are in air changes per hour (ach). All three parameters have to be estimated or measured. Calculations were made using the equilibrium form of the mass-balance equation and assume a fixed emission rate of 26 mg/m^3 of RSP.

Shaded area shows the range of RSP emissions that could be expected for a residence with one smoker smoking at a rate of either 1 or 2 cigarettes per hour for the range of mixing, ventilation, and removal rates occurring in residences under steady-state conditions.

Source: NRC, 1986.

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17.
Experiments on healthy young adults exposed to secondhand smoke show higher resting heart rates, higher blood carboxyhemoglobin, a significant reduction in the amount of oxygen that can be absorbed during exercise, and a shorter time to exhaustion when running on a treadmill.

pp. 8-9

18.
Patients exposed to secondhand smoke who have preexisting myocardial infarction have more ventricular arrhythmias (irregular heart beats) during passive smoke exposure than when breathing clean air.

page 9

19.
Other studies comparing the exercise ability of healthy individuals to people with heart disease found that exposure to secondhand smoke lengthened the time to recover resting heart rate at the end of exercise, ...

page 9

20:
Passive smoking also significantly increased the amount of a chemical called lactate in venous blood, ... (27).

page 9

27. McMurray, R.G., Hicks, L.L., Thompson, D.L. The effects of passive inhalation of cigarette smoke on exercise performance. Eur J Appl Physiol 1985;54:196-200

16.
People with coronary heart disease as well as normal people cannot exercise as long or reach as high a level of exercise after breathing secondhand smoke than when breathing clean air.

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